

greatly. The course of the disease is often progressive over a number of months, death being due to respiratory failure and occasionally to cor pulmonale or infection with opportunistic organisms, e.g. *Nocardia*.<sup>3</sup>

The case described illustrates many features of this disease. However, asymptomatic involvement is also recorded.<sup>4</sup> In our patient this disease was not suspected because pulmonary alveolar proteinosis is rare; lung biopsy established the diagnosis.

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# Giant intracranial aneurysm

## A case report

B. STOCKLAND

### Summary

A 27-year-old woman presented with a history of loss of balance and deafness in the left ear for 2 weeks. Computed tomography and arteriography demonstrated a giant aneurysm 49 mm in diameter in the posterior cranial fossa. Surgical removal of the aneurysm was unsuccessful.

*S Afr Med J* 1986; 70: 693-694.

Giant intracranial aneurysms have been well documented in both radiological and neurosurgical journals. This case is presented because it demonstrates the biggest cerebral aneurysm encountered at this hospital to date and also underlines the fact that these aneurysms can present as space-occupying lesions and mimic intracranial tumours. Mistaking such an aneurysm for a tumour can become a nightmare if the patient is subjected to surgery. The case is also of interest in that the aneurysm contained no thrombus, and aneurysms of this size are usually either partly or completely thrombosed.

### Case report

A 27-year-old white woman presented with a history of loss of balance for 2 weeks, which had become progressively worse during the preceding 4 days. She also complained about deafness of recent onset and vomiting for the preceding 2 days.

On examination she was fully conscious, co-operative and well-orientated. She had mild dysarthria and sensorineural deafness in the left ear. Vertical and horizontal nystagmus was present. Rom-

berg's sign was not present but the patient was ataxic and demonstrated loss of balance on the left side when walking. Motor and sensory systems were otherwise intact. She was mildly hyper-reflexic. Her cardiovascular system was normal — a pulse rate of 64/min, a blood pressure of 120/80 mmHg. The lungs were clear and both the gastro-intestinal and urogenital systems were normal. Routine biochemistry test results were negative. Plain skull radiographs showed pressure erosion of the clivus but no pathological calcification or evidence of raised intracranial pressure.

Computed tomography (CT) showed posterior displacement of the brainstem with compression of the fourth ventricle and obstruction of the ventricular system causing dilated third and lateral ventricles. The posterior displacement of the brainstem was caused by a well-defined mass of increased density with no surrounding oedema. After intravenous injection of 50 ml Conray 420 the mass was strongly enhanced and the basilar artery was visible next to the mass, but displaced to the right side (Fig. 1).

At this stage a tumour in the posterior fossa, possibly a meningioma, was suspected, although the possibility of an aneurysm was also raised. The four-vessel cerebral angiogram demonstrated a large aneurysm measuring 49 mm in diameter arising from the superior region of the basilar artery, at or close to its bifurcation into the posterior cerebral arteries. The aneurysm displaced the basilar artery to the right side. The only blood flow to the posterior fossa that could be demonstrated angiographically was via poorly filling posterior inferior cerebellar arteries (Fig. 2).

At surgery the full extent of the aneurysm and the position of its neck could not be identified. The patient died postoperatively after respiratory arrest, possibly as a result of disturbance of the brainstem. No autopsy was performed.

### Discussion

Giant intracranial aneurysms are rare. Aneurysms are classified as giant aneurysms if they are 2.5 cm or more in diameter. They are found more commonly in the posterior intracranial circulation.<sup>1</sup> In one series, 62% of the aneurysms were located in the posterior circulation, 24% being at the bifurcation of the basilar artery.

In the past intracranial aneurysms have been thought to rupture only rarely. More recent evidence suggests that rupture is not uncommon, especially in children.<sup>2</sup> Furthermore, 20% of

Department of Radiology, University of Stellenbosch and Tygerberg Hospital, Parowvallei, CP

B. STOCKLAND, M.B. CH.B., M.MED. (RAD. D.)

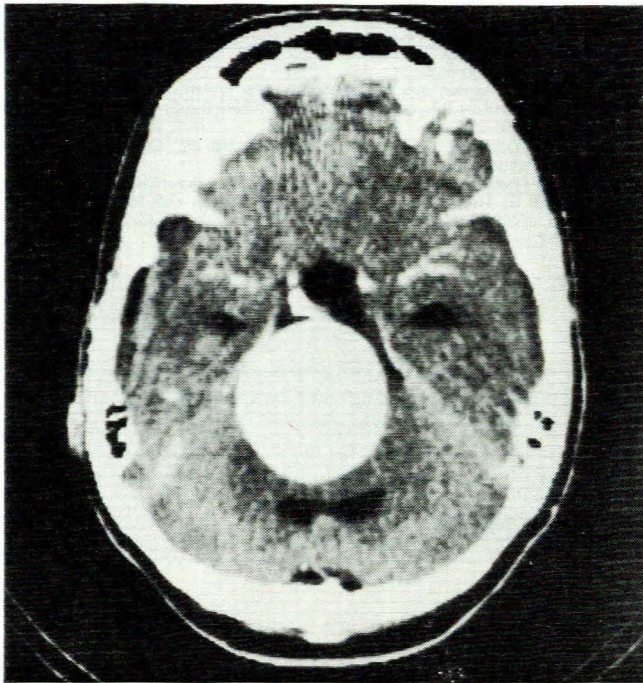


Fig. 1. Contrasted CT of the brain shows the dense enhancement of the aneurysm with a sharp, well-defined edge.

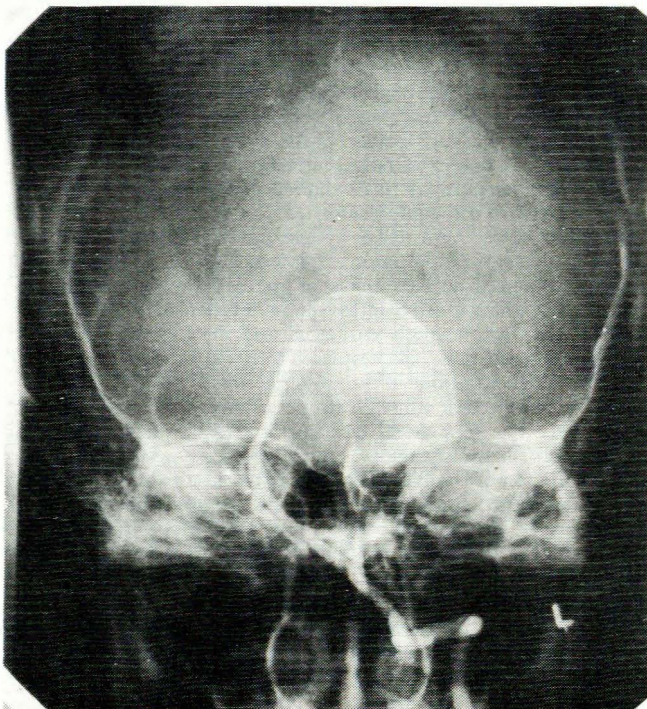


Fig. 2. Early anteroposterior arteriogram of the vertebrobasilar system showing both filling of the aneurysm and the gross displacement of the basilar artery.

patients have other intracranial aneurysms. Although these patients may present with a history of vascular headaches, the more usual presentation is that of an intracranial space-occupying lesion. Cranial nerve damage occurs frequently, most commonly in VIII followed by V, VI and IX in order of frequency.<sup>1,3</sup> Nystagmus as well as pyramidal tract lesions may occur. These aneurysms may increase in size if left untreated. A review of the literature by Artmann *et al.*<sup>4</sup> identified 130 cases of enlarging intracranial saccular aneurysms; 22 of these were giant aneurysms.

Radiological investigation should include plain films of the skull, which may show evidence of raised intracranial pressure, curvilinear calcification in the aneurysm if it is partly or completely thrombosed, and evidence of bone erosion. The only plain film finding in the case discussed here was erosion of the clivus.

After the injection of contrast medium, three different CT patterns have been described in giant intracranial aneurysms:<sup>5</sup> (i) a thin-walled aneurysm may present as a well-defined dense mass, which is strongly enhanced after contrast injection; the homogeneous enhancement may, as in the case described here, simulate a meningioma; (ii) a partly thrombosed aneurysm may appear as a central or eccentric zone of mildly increased density (representing the lumen), surrounded by a isodense zone (the thrombosed portion), and peripheral to this a zone of increased density (representing a layer of fibrous tissue), all of which form a 'target' sign; and (iii) a completely thrombosed giant aneurysm may appear as in (ii), but the central zone of enhancement is absent.

Angiography should follow CT to confirm the diagnosis and prevent misdiagnosis of the lesion as a solid tumour such as a meningioma. Angiography will show only the non-thrombosed areas of the aneurysm. Its actual size is estimated by evaluation of the degree of displacement of surrounding vessels.

The prognosis in both treated and untreated patients remains poor.<sup>6</sup> Morley and Barr<sup>7</sup> had 28 cases in their series; 14 of these patients were left untreated. Of these, 4 died as a result of subarachnoid haemorrhage and 1 due to a posterior fossa mass effect. Bull<sup>8</sup> reported 12 cases which were treated surgically; 5 of these patients died.

It is clear that giant aneurysms must be considered in the differential diagnosis of space-occupying lesions, especially those in the posterior fossa.

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